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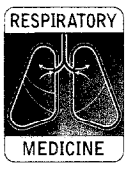

Case Reports

Post-traumatic pleural effusion: demonstration of local complement consumption

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The present case report describes a 27-year-old patient who presented with post-traumatic pleural effusion. Analysis of the pleural fluid showed hypereosinophilia (990 mm^{-3}), a decreased level of total complement, and decreased levels of C3 and C4 fractions (less than 50% of normal serum levels), indicating a local consumption mechanism for complement. Complement serum levels (CH50, C3, C4) were normal. All other aetiologic possibilities were eliminated.

This case suggests that the immunopathological mechanism of post-traumatic pleural effusion may involve activation of the classical pathway of complement and a recruitment of inflammatory cells such as eosinophils.

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Introduction

The physiological mechanisms of post-traumatic pleural effusions are unknown. Various authors have found pleural and serum hypereosinophilia which could be the result of an immune complex reaction (1–3). However, to the authors' knowledge, the levels of complement proteins have not been measured in any previous cases of hypereosinophilia. The present case report describes a young patient who presented with a post-traumatic pleural effusion, in which the difference in serum and pleural levels of total complement and the C3 and C4 components evoked their local consumption.

Case Report

A 27-year-old patient was admitted to the authors' clinic in March 1991 with left pleural effusion. He was addicted to heroin and benzodiazepines; he was a smoker (5 pack-years) and gave a history of a left tibial fracture in 1982. In December 1990 following a left thoracic trauma, he had left-sided chest pain. In February 1991, a chest X-ray was performed because of acute bronchitis and persistent left-sided chest pain, which showed left pleural effusion (Plate 1). There was no regression of the pleural effusion, despite antibiotic treatment. He was hospitalized in March 1991 to investigate this pleural effusion further.

Laboratory analysis of his blood showed an absolute eosinophilic count of 900 mm^{-3}. There were signs of moderate inflammation, with an erythrocyte sedimentation rate of 27 mm at 1 h and 66 mm at 2 h, a fibrinogen level of 5·6 $g\,l^{-1}$ and a c-reactive protein level of 29 $mg\,l^{-1}$. Hepatic and renal functions were normal. Antinuclear antibodies, circulating immune complexes and cryoglobulin were absent. Pulmonary ventilation and a perfusion scintigraphy scan were normal. A total of 300 ml of serohaematic exudative pleural fluid was obtained by aspiration. It contained 16% neutrophils,

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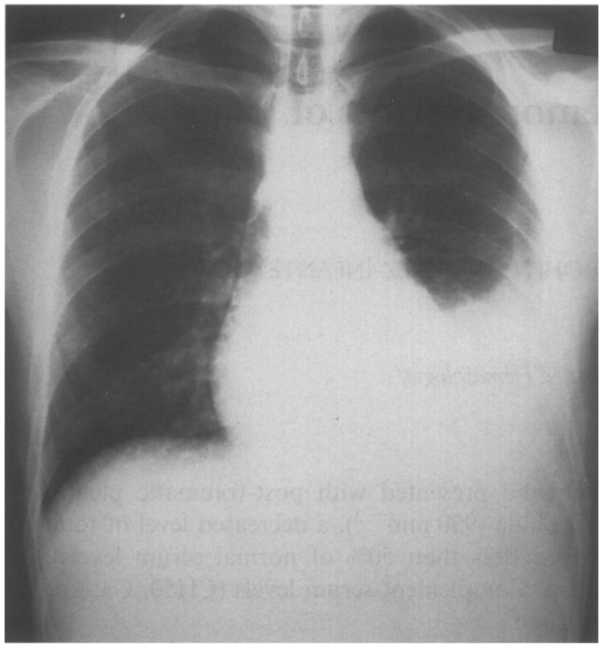

PLATE 1. Chest X-ray at the patient's admission.

6% lymphocytes and 66% eosinophils. Pleural fluid proteins were 41 $g\,l^{-1}$ and glucose 6·8 $mmol\,l^{-1}$. The pleural fluid to serum lactate dehydrogenase ratio was 3:1. There were neither *Mycobacterium tuberculosis* nor malignant cells on direct examination.

Total complement haemolytic activity assay (CH50) (4), and C3 and C4 levels (measured by Nephelometry Laser, BNA Behring) in peripheral blood and pleural fluid were measured twice over a period of 8 days, and are shown in Table 1.

The patient was treated with oral antibiotics (penicillin A), mucolytics and chest physiotherapy. However, he left the hospital against medical advice. A chest X-ray performed in June 1992, 15 months after his prior admission, showed total regression of the pleural effusion (Plate 2).

Discussion

The diagnosis of post-traumatic pleural effusion is guided by the history of trauma to the chest wall, with clinical signs of pleural effusion appearing secondarily. Absence of rib fracture does not rule out this possibility. Campbell *et al.* (1) reported cases of post-traumatic pleural effusion without rib fracture. This can also be diagnosed by a process of elimination, as in the present case, using clinical examination, laboratory examination of peripheral blood and pleural fluid, a pulmonary scintigraphy scan and radiological evolution to eliminate all other aetiological possibilities of pleural effusion, such as pulmonary embolism, malignancy, systemic diseases and empyema.

The patient's pleural fluid had two characteristics: hypereosinophilia, which is a classical phenomenon in this kind of pathology; and local consumption of complement. This combination has not been reported previously. It has been reported that eosinophilic accumulation could be a response to the presence of immune complexes. Laster and Gleich (2) reported the presence of a chemotactic activity elicited by the IgG and IgM aggregates with the presence of complement. Cohen and Ward (3) reported the intervention of a chemotactic factor of

TABLE 1. Serum and pleural complement levels

	Serum complement			Pleural complement		
	CH50* (UI ml^{-1})	C3† ($g\,l^{-1}$)	C4† ($g\,l^{-1}$)	CH50 (UI l^{-1})	C3 ($g\,l^{-1}$)	C4 ($g\,l^{-1}$)
13 January 1991	51·5	0·87	0·22	20·5 (0·43)‡	0·35 (0·49)‡	0·086 (0·33)‡
21 January 1991	39	0·76	0·20	16 (0·47)‡	0·34 (0·47)‡	0·095 (0·36)‡
Normal serum levels	47 ± 12	0·71 ± 0·19	0·26 ± 0·13			

*Haemolytic assay.
†Laser nephelometry, BNA Behring nephelometer.
‡Values in parentheses correspond to ratios of normal serum levels of CH50, C3 and C4.

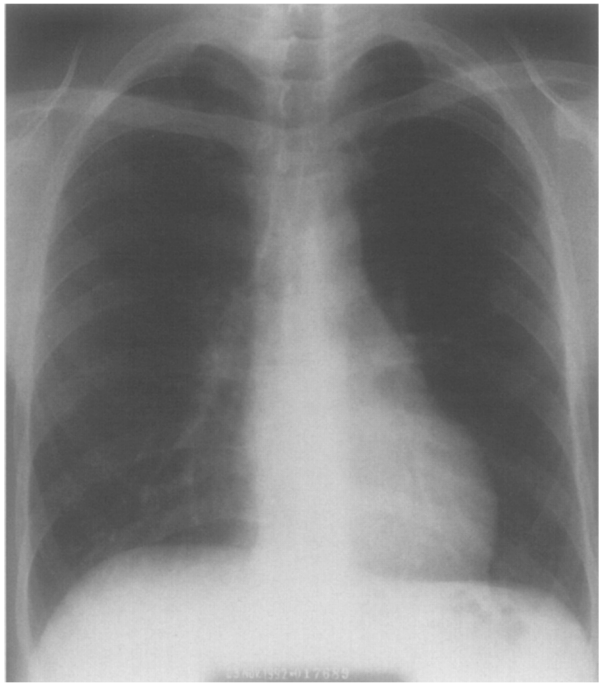

PLATE 2. Chest X-ray 15 months after hospitalization.

eosinophils produced by a specific interaction of lymphocytes and immune complexes.

Local consumption of pleural complement in pleural effusions has been shown by several authors to be associated with rheumatoid arthritis and systemic lupus erythromatosus. In the study by Glowsky *et al.* (5), C3, C4 and CH50 values in pleural fluid were less than 50% of blood values. Similarly, Miguéres *et al.* (6) have shown local consumption of total complement and of the C3 fraction in purulent pleural effusion. The present patient's exudative pleural fluid CH50, C3 and C4 levels varied between 33 and 47% of the mean theoretical values in blood. In contrast, in lung cancer, pleural effusion and infections, pleural complement levels have been found to be greater than 50% of normal serum values. In two cases of rheumatoid arthritis presenting with pleural effusion, Pauli *et al.* (7) reported that the pleural fluid complement levels were less than 10% of normal blood values. A concomitant decrease of pleural fluid C3, C4 levels has been reported in patients with rheumatoid and lupus erythromatosus pleural effusion. However, Glowsky *et al.* did not find a decrease in the C3 fraction in such patients (5)

In summary, the local consumption of complement as observed in the present patient could be similar to that found in rheumatoid arthritis and systemic lupus erythromatosus. This phenomenon could be related to the presence of immune complexes in the pleural fluid to the pericardial and synovial effusions found in these two systemic diseases (8,9).

This finding suggests that the post-traumatic pleural effusion in the present patient could be the result of an immunological mechanism in which immune complexes' immunoglobulin fractions activate the classical complement pathway and recruit inflammatory cells such as eosinophils.

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